operation which was temporarily successful, but the child died in two weeks from pneumonia, so that he did not know the late results. It was done when the child was but a few months old, which is the best time to operate.

(*To be continued.*)

NEW YORK NEUROLOGICAL SOCIETY.

May 1, 1906.

The President, DR. JOSEPH FRAENKEL, in the Chair.

The Chronic Progressive Softening of the Brain. Report of Cases with Autopsies Simulating Cerebral Tumor.—By Dr. J. Ramsay Hunt. The author reported in detail two such cases coming under his observation, and gave the autopsy findings. Case I was of special interest because of the clinical resemblance to brain tumor. The apoplectiform seizure which occurred one year before the appearance of any focal symptoms was of very doubtful nature; there was no paralysis at the time, and the recovery was absolute and complete. After the beginning of focal symptoms in the right side of the face, the course was gradual and progressive, appearing next in the fingers, then in the hand and forearm, and later only slightly in the leg. During this entire period and even antedating the focal symptoms, severe paroxysmal headaches, usually unilateral, were present, with some variable tenderness over the left parietal region. The absence of optic neuritis had given rise to serious doubts as to the real nature of the lesion, but this, as was well known, by no means excluded tumors, especially those situated in or near the cortex. As all the internal measures employed had failed, and as the condition was slowly progressing, surgical exploration seemed not only justified, but imperative. The ill-effects of the surgical measures were to be attributed to exploratory punctures with an aspirating needle, in search of a possible cyst. In diseased and softened brain tissue, aspiration, no matter how delicately performed, was not without danger, and was liable to induce hemorrhagic extravasations, which broke down and formed favorable foci for infection. Furthermore, there was no question that the dangers of infection, even with all modern aseptic precautions, were much more imminent when a softened, ill-nourished area of the brain was the seat of exploration. That danger should always be borne in mind when the cerebral cortex was exposed and found normal, and the question of further exploratory measures arose.

The usual sequence of symptoms in this case, so unlike that of softening in general, was to be referred to the parietal arteriosclerosis at the junction of the internal carotid, middle cerebral and posterior communicating arteries, with occlusion of the central perforating at their point of origin from the main trunks. This lesion had induced a gradual and successive obliteration of these central perforating end arteries, producing the progressive and insidious development of hemiparesis and hemiparesthesia, with objective sensory disturbances. In brief, the picture of the so-called chronic progressive softening of the brain.

In Case II, while the lesion had been progressive, involving first the right leg, then the right arm, and later the left leg, the development was by no means so constantly or uniformly progressive as in case I. The mode of onset, and the acute exacerbations suggested rather a vascular process than a neoplasm. As the symptoms in cases of vascular tumors not uncommonly suffer a sudden increase in the focal manifestations from hemorrhages, and as optic neuritis with beginning atrophy was present, the possibility of tumor in the region of the corpus callosum, invading the Rolandic area on the two sides, was for a time given serious consideration. The marked inertia, extreme apathy, and mental enfeeblement tended to favor that interpretation; but as the focal symptoms failed to progress materially during eight months' observation, a generalized arteriosclerosis and encephalitis came to be regarded as the probable underlying cause, and the optic neuritis was referred to the glycosuria which was present, or possibly, as was rarely the case, was dependent upon arteriosclerosis of the vessels of the optic nerves.

Pathologically, the case was of interest from the limitation of the obliterating thrombotic process to the central perforating arteries passing to the internal capsule and basal ganglia. The main trunks of the circle of Willis were singularly free from sclerosis, and the thrombi contained in the middle cerebrals were of quite recent origin. More difficult of interpretation was the extensive breaking down of the walls of the lateral ventricles. That process could not be regarded as an encephalomalacia in the ordinary acceptation of the term, as there was total absence of the usual histological changes accompanying such softening; viz., myelin degeneration and granule cells. Only minute hemorrhages and red blood cells were present in the ragged margin surrounding the ventricles, which would seem to indicate that we were not dealing with a post-mortem change or artifact. The periventricular localization of the breaking-down process was also unusual, and suggested an etiological relation to the contents of the lateral ventricle. The influence of diabetes and the tendency which induced to tissue gangrene also arose as a possible predisposing or etiological factor.

In general, Dr. Hunt said, the clinical picture presented by his cases was that of a progressive and gradually developing hemiplegia, and the character of the general cerebral symptoms resembled those described by Wernicke as chronic progressive softening, without evident vascular lesions. The main trunks of the vessels of Willis were free from occlusion, but histological study demonstrated the obliteration of the central end arteries in the ganglionic and capsular regions. It was readily conceivable how such central arteriosclerosis, with occlusion, would not only produce degeneration in the distribution of the terminal end arteries obliterated, but might also determine more extensive areas of softening in that portion of the white substance in the centrum ovale, where the cortical and central circulation met, and which represented an area of diminished nutritional resistance. It therefore seemed that the existence of a progressive softening not dependent upon vascular disease of the type described by Wernicke might be fairly questioned; certainly until demonstrated by a case subjected to modern laboratory methods.

In addition to central arteriosclerosis as a cause of chronic progressive softening must be added constriction and obliteration of the carotid arteries in the neck.

The clinical importance of this subject, Dr. Hunt said, lay in the possible resemblance to abscess, encephalitis, and tumor of the brain.

Dr. Joseph Collins said he had recently devoted considerable attention to this subject, and the only criticism that he had to offer of Dr. Hunt's paper was relative to its title. He did not think these cases should be called instances of chronic progressive hemiplegia without stating that they were of arteriosclerotic origin, and he believed that the most appropriate name under which they could be classified was cerebral arteriosclerosis. From a careful review of the literature on the subject, he had no hesitancy in stating that all the cases of so-called chronic progressive hemiplegia were of arteriosclerotic origin.

In connection with these cases, Dr. Collins said, we should not fail to appreciate the fact that while in many instances we may have to deal with a generalized arteriosclerosis, there were others in which the arteriosclerotic changes were strictly localized to certain viscera or sections of viscera, the blood vessels in other sections of the body being entirely free from manifestations of the disease. Cases were not uncommon in which the symptoms were indicative of arteriosclerosis, and where the lesions, both macroscopically and microscopically, were apparently confined to the smaller vessels of the brain, while the basilar and larger arteries were entirely free from any evidences of the disease.

Dr. Collins said that in the May, 1905, issue of *The Post-Graduate,* he had published an article under the title of "A Case of Cortical Cerebral Arteriosclerosis, with Distinct Focal Symptoms," which reported in detail the history of a hard-working married woman, fifty years old, whose symptoms, as well as the autopsical record, parallelized, in almost every item, those which Dr. Hunt had presented. The symptoms in his case, Dr. Collins said, seemed to indicate that the lesion was a generalized arteriosclerosis, with focal manifestations in the left motor cortex, but there was such diversity of opinion among the physicians who saw her, several of whom made the diagnosis of hysteria, that the subsequent history of the case proved both useful and interesting.

In speaking of the differential diagnosis between brain tumor and cortical cerebral arteriosclerosis, Dr. Collins said he thought the occurrence of progressive hemiplegia in an individual over fifty years old had no more merit as a diagnostic feature of brain tumor than a dozen other symptoms that might be mentioned. On the contrary, that symptom, occurring in an individual over fifty, should excite the suspicion of cortical cerebral arteriosclerosis.

In concluding his remarks, Dr. Collins again emphasized the fact that arteriosclerotic conditions might be either local or general, and that in the proper recognition of their effects, splanchnic arteriosclerosis must be entirely divorced from arteriosclerosis of the brain.

The President, Dr. Fraenkel, said that the second case reported by Dr. Hunt was under his observation for a time, and he regarded it as one of brain abscess. The symptoms pointed to such a lesion, and the postmortem explanation of the case was not quite satisfactory. The accumulation of broken-down material could very well explain the clinical impression of abscess. Dr. Hunt's paper was of great importance in calling attention to arteriosclerosis as a factor of progressive hemiplegia.

Dr. Hunt, in reply to Dr. Collins, called attention to the fact that he had reported his cases under the title of chronic progressive softening of the brain; not as cases of chronic progressive hemiplegia.

A Case of Huntington's Chorea.—Presented by Dr. Edward D. Fisher. The patient was a man, thirty-five years old, who gave a family history of this disorder, his father and an older brother having died of the same disease. In the case shown, the symptoms had first manifested themselves about three years ago. They had progressed steadily, and assumed a choreiform type. Speech was affected, but there was no disturbance of mentality. The reflexes were exaggerated. There was no pain. A younger brother had recently shown the initial stages of this same disorder.

Dr. J. F. Terriberry said that Dr. Fisher's case recalled a similar instance in a family which he had observed for a number of years, in whom three male members were victims of this disease. Two had died, while the third was still mentally clear and able to work, although his condition was very distressing. Attempts at talking were accompanied by exaggerated movements of the face and extremities.

A Case of Arteriocapillary Fibrosis Localized in the Medulla, and Clinically Resembling Myasthenia Gravis.—By Dr. Harlow Brooks. The patient was a male, forty-three years old, born in Russia, and for twelve years a resident of the United States. His occupation was that of a photographer. His father died from diabetes, which terminated in "brain fever." His mother died from pneumonia. He did not know how many brothers and sisters he had, but remembered that a great many of them died. Four, including the patient, were still alive, and, with the exception of himself, were in good health. There was no history of malignant, tubercular or syphilitic taint.

The patient's present illness began four years ago. He first noticed that his fingers became weak while holding a photographic plate, which finally fell from his grasp. When he stooped to pick up the plate he could scarcely raise himself upright; he apparently lost control of the left arm and leg, and barely managed to crawl to the adjoining room, where he was put to bed. He did not remember what happened to him after that for fourteen days. After that attack he was regularly treated by a physician, who employed drugs and electricity. Under that treatment he gradually regained control of the paralyzed extremities, and after some months he was able to return to his work. In August, 1904, he began to feel very weak and could not stand upright. He had no pain; no convulsions, but his statements in that regard were somewhat conflicting.

Upon admission to the Montefiore Hospital the patient complained that he felt weak. He could not stand upright. There was no pain nor dizziness. He complained of double vision. There were no enlarged glands. The expression of the face was pathetic, dull and listless. Innervation of face normal. No nystagmus, and the pupils reacted to light, pain and accommodation. Ears normal. The abdominal and thoracic viscera were apparently normal, with the exception of a rough, systolic murmur. The knee-jerks were apparently diminished. The tendo Achilles reflexes were present. The Babinski reflex was absent.

On Feb. 13, 1905, the patient was allowed out of bed. He complained of nothing in particular. On June 29, after a warm bath, he began to complain of dyspnea, which, in spite of cupping and other treatment, gradually became more severe. Moist, gurgling râles were heard over the upper portions of the lungs. Death occurred on the following day.

After reporting in detail the post-mortem and pathological findings, Dr. Brooks said that clinically this case seemed to correspond quite closely to the picture of myasthenia gravis pseudoparalytica, the presence of the symptom of muscular tiring being very well marked, particularly in the muscles of the speech. Furthermore, the myasthenic reaction of Jolly appeared to have been present, although the speaker said he understood that that reaction was by no means diagnostic. No macroscopic muscular atrophies were present.

Pathologically, the case was clearly not one of myasthenia gravis pseudoparalytica, since lesions were found which abundantly explained all the symptoms present, and even the cause of death. Muscular atrophy, though of but limited degree, was present, and the medullary changes were pronounced, though a very careful gross examination, in which particularly minute note was made of medullary conditions, failed to demonstrate any morphological alterations, and only close microscopic study demonstrated the vascular alterations which abundantly explained all the symptoms.

The vascular disease was clearly allied to arteriofibrosis, and the remarkable feature of this lesion was its markedly limited distribution to the trunks of the medulla almost exclusively. It was of course manifest that the symptoms appearing four years before the onset of the complaints which the patient presented on entrance to the hospital were due to cerebral hemorrhage.

In attempting to discover an etiological factor for the explanation of these pronounced vascular changes in so young a subject, Dr. Brooks said he did not think we had far to look. It had been abundantly demonstrated that excessive use of tobacco produced arterial disease of marked degree; nicotine in itself was one of the most active vaso-constrictors, and it seemed highly probable that the excessive use of tobacco in this case was productive of those vascular changes, chiefly confined to the medulla, which gave origin to the symptoms indicative of myasthenia gravis.

Dr. B. Sachs said the subject of myasthenia gravis pseudoparalytica was a very difficult one, and he thought it was still an open question whether or not there was an anatomical lesion underlying the disease. Some had reported the presence of such a lesion, while others had doubted it, and while that aspect of the subject was still *sub judice*, the least one could do was to clearly establish the clinical picture of the disease.

In the case reported by Dr. Brooks, the speaker said, he failed to see any clinical grounds upon which to base the diagnosis of myasthenia gravis pseudoparalytica. While the patient had some bulbar symptoms, there were no autopsical findings to account for them. All that we had a right to say was that it was a case with more or less pronounced bulbar symptoms, and such cases were not uncommon. Years ago, Westphal reported a similar case, without anatomical findings.

Dr. Fraenkel said he wished to confess that he was the first one to suggest the diagnosis of myasthenia gravis pseudoparalytica in the case reported by Dr. Brooks. The case had been presented to him as one of hysteria or neurasthenia. He could not accept either of those diagnoses, and from the absence of objective symptoms at the time of examination, and the disproportion between the extent of the medullary symptoms at the time of the seizures and the lack of such symptoms during the intervals, he thought a diagnosis of myasthenia gravis pseudoparalytica was justified. He conceded that it was not a typical example of that affection, and thought it possibly represented a transitional stage.

Dr. Joseph Collins said he regarded Dr. Brooks' paper as a most important contribution to the pathology of the central nervous system. It described a transitional stage between cases that were apparently without anatomical foundation, and others in which the anatomic conditions had been demonstrated. The speaker said that in his opinion, however, Dr. Brooks' case had been reported under a misnomer, and so far as he could see, there was no justification for the diagnosis of myasthenia gravis pseudoparalytica. A dozen characteristic symptoms of that disorder could be enumerated that were apparently entirely absent in Dr. Brooks' case. Why was it necessary to take a case that presented all the symptoms of bulbar paralysis, such as Erb had recently described, and call it myasthenia gravis pseudoparalytica? A similar case had recently been reported to the society by Dr. Edwin G. Zabriskie and himself. The speaker said he now had the records of five cases of spinal arteriosclerosis in which the general arteriosclerosis was not pronounced, but the changes in the cord were profound. In one of them the clinical picture was that of Landry's paralysis. He ventured to say that Dr. Brooks should have reported his case under the head of arteriosclerosis of the oblongata, producing the clinical features of asthenic bulbar paralysis.

Dr. Terriberry said that most of the symptoms of myasthenia gravis pseudoparalytica were absent in the case reported by Dr. Brooks, while a number of the symptoms mentioned were not present in that disorder. The clinical picture corresponded more closely to that due to a weak and flabby heart; the acute crises, with edema of the lungs, could thus be readily understood. That view was also supported by the post-mortem findings, the heart being large, its muscles flabby, and its vessels diseased. As we had no knowledge of the pathological substratum of myasthenia gravis pseudoparalytica, and as a most careful and thorough autopsy revealed a more or less general arteriosclerosis, he believed that was the underlying factor in the case, and that the diagnosis as given could not be maintained.

Dr. Brooks, in closing, said he was positive that the cause of death in the case he had reported was not myocarditis; that fact was perfectly obvious to all who saw it. There was no generalized arteriosclerosis, and a minute examination of the viscera showed a perfectly normal set of arteries.

The Surgical Treatment of Trigeminal Neuralgia. A Study of the Causes of Recurrences After Operative Treatment, with Suggestions as to the Best Methods of Obviating Post-Operative Recurrences.—Dr. Alexis V. Moschcowitz read a paper on this subject, in which he offered the following conclusions:

1. Eliminate any possible etiological factors, such as tumors, carious teeth, antral disease, malaria, syphilis, etc.

2. Determine accurately the branch or branches that are involved.

3. The operation should be performed as near to the periphery as possible.

4. The operation should be performed early. This is important, because the earlier the case the more chances there are that a peripheral operation will be of benefit.

5. Whatever the character of the operation may be, the dominant principle must be the *prevention of regeneration* of the affected nerve.

More specifically, the operations may be classified under two headings, depending on the nerve or nerves affected.

(a) Peripheral operations: If the supraorbital, infraorbital, mental, malar, or inferior dental branches, either singly or collectively, are involved, the operation consists in division of the nerve, and plugging up the foramen by a gold or silver button or wire.

(b) Central operations: If the neuralgia involves the upper teeth and palate (superior maxillary division), or the tongue (inferior maxillary division), existing either singly or together with the other nerves described above, the operation as outlined by Abbe must always be performed, substituting, however, celluloid or a gold button instead of rubber tissue.

6. Finally, if the above principles of treatment of trigeminal neuralgia are carried out, Dr. Moschcowitz said he believed that the operation of extirpation of the Gasserian ganglion would become entirely unnecessary.

Dr. F. Kammerer said he fully agreed with Dr. Moschcowitz that the mortality of the operation of extirpation of the Gasserian ganglion for trigeminal neuralgia was high, and furthermore, it would be much higher if all the cases had been recorded. The speaker said that personally he could add two cases to the list of fatalities following this operation. In one, death resulted on the second day from hemorrhage, and in the other from infection on the fourteenth day after the operation. In the last case, severe hemorrhage after evulsion of the ganglion necessitated the use of a tampon.

Dr. B. Sachs said that if Dr. Moschcowitz had any substitute to offer for the very severe operation of extirpation of the Gasserian ganglion, it was something that was very much to be desired. The speaker said he had seen seven or eight of these operations done, and he knew of no other operation that had impressed him so unfavorably, not only because of its extreme difficulty and severity, but also on account of the uncertainty as to whether the ganglion had really been extirpated or not. In some of the cases he had observed, the sensibility of the affected side of the face had not been entirely abolished by the operation, which could be explained either on the ground that there was some anomaly of the nerve to begin with, or else that the ganglion had not been completely extirpated. Dr. Moschcowitz had reduced the problem to a very simple form, and the speaker said he only hoped that the solution he offered would prove correct. By interposing a foreign body between the cut ends of the nerve, or plugging its point of exit, we could be tolerably certain that there would be no actual reunion, but whether there might not be sufficient regeneration to allow certain of the nerve fibres to pass around the foreign body and unite was another question.

Dr. William M. Leszynsky said that one rather suggestive remark by the reader of the paper was that these patients should submit to early operation. The speaker said that within the past ten years he had not seen a single patient with trigeminal neuralgia of less than one year's duration in whom improvement or cure had not followed suitable non-surgical treatment. Various methods of relief were at our command before surgical intervention should be even suggested. Many of these patients who had never submitted to a systematic course of treatment were operated on unnecessarily. Furthermore, that even after operation on the nerve branches, as ordinarily performed, the nerve would ultimately regenerate.

Dr. Leszynsky said that within the past three years he had seen four cases of severe trigeminal neuralgia cured by non-surgical methods. One of the patients was an old lady, seventy years of age, who had ninety paroxysms of tic douloureux in twenty-four hours, and who completely recovered under medical treatment. She has been entirely free from pain for several years. In her case, an operation had been recommended by several surgeons. In conclusion, Dr. Leszynsky said that of course operation was necessary in some patients, but he thought that the percentage of cases of trigeminal neuralgia in which an operation was indicated was very small.

Dr. Edward D. Fisher said that doubtless the cases that Dr. Moschcowitz had in mind in his paper were those that had failed to respond to the usual methods of treatment. His usual plan in regard to operative measures was to first advise a resection, which would give the patient at least temporary relief, and then later on, if it became necessary, the more severe operation could be done. The operation suggested by Dr. Moschcowitz appeared feasible; it would certainly give the patient a longer interval of relief than any of the well known conservative measures, and was free from the danger of the more radical ones.

Dr. W. Sohier Bryant said that in the light of recent studies, he thought that many cases of trigeminal neuralgia were of reflex origin, and that a certain number might be relieved either by proper investigation and treatment of disease of the antrum or the other accessory sinuses, or by relieving intranasal pressure.

Dr. Moschcowitz, in closing, said that by advising early operation, he did not mean to imply that it should be done before a thorough trial of non-surgical methods of treatment had been given. More particularly, however, by advising early operation he wished to emphasize the fact that this type of neuralgia usually began in one of the smaller branches of the trigeminal nerve, and the cases should be operated on before the larger branches became involved.

Dr. Moschcowitz said the comparatively few cases of trigeminal neuralgia he had seen had been sent to him by neurologists; a point, which, to his mind tends to prove that internal medication has been tried to a sufficient extent.

Functional Derangement of the Ears and Upper Air Tract in the Insane.—Dr. W. Sohier Bryant, who read this paper, said the observations upon which it was based were undertaken for the purpose of showing the relation, if any there might be, between insanity and functional derangement of the ear and upper air tract. In order to err on the conservative side, when there was doubt in the results of an observation, it was classified as normal, thus giving the weakest possible showing for the relationship of ear disease, tinnitus and hallucinations of hearing.

One hundred and sixty-one patients were examined at the Manhattan Hospital. Most of these cases were chosen on account of their hallucinations of hearing. The rest were taken by accident without any special plan. The psychiatric classification was as follows: Dementia præcox, 63; paranoia, 20; dementia paralytica, 20; alcoholic insanity, 19; mania depressens, 16; senile dementia, 14; epileptic insanity, 6; various, 3.

The ear classification was as follows: Otitis media catarrhalis, 71; otitis media purulenta, 38 (active or cicatrized); foreign bodies, 14; labyrinthine or nerve disease, 23. In ten of these, the diagnosis was definitely established. In thirteen the diagnosis was not positively differentiated from an adhesive process in the middle ear. Among the 161 cases, fifteen were free from ear disease.

There were hallucinations of hearing in 134 cases; in 120 of these, they were associated with ear disease; in fourteen there was no ear disease. Of the latter, five had tinnitus, and that symptom was present in 63 of the entire number. There were no hallucinations of hearing in 27 cases, and of that number, 25 had ear disease. Among the 161 cases examined, 146 had ear disease, and 15 were free from ear disease. Only three cases out of 161 had perfectly normal hearing.

Naso-pharyngeal conditions: Total or partial obstruction of the passage, 79; obstruction above the lower turbinate, 15; hypertrophic conditions without obstruction, 37; atrophic conditions, 5; normal, 25.

Naso-pharyngeal and aural diseases in their relation to insanity, Dr. Bryant said, might be classified under five heads: (1) Naso-pharyngeal and aural disease which had no relation to the co-existent psychical dis turbances. (2) Naso-pharyngeal and aural disease consequent to insanity. (3) Naso-pharyngeal and aural disease due to the same cause as the co-existent insanity. (4) Naso-pharyngeal and aural disease which were a determining factor in the symptoms of the psychosis. (5) Naso-pharyngeal and aural disease which were the exciting cause of the insanity

The prognosis for psychical improvement from treatment of the nasopharynx in the cases where the nasal complication was a disturbing factor was good. It was also good when active aural disease was a disturbing factor. The prognosis was bad where chronic, inactive aural disease was the disturbing factor. Those cases were difficult to treat even with the co-operation of the patient. Without that co-operation, they were still more difficult.

Dr. Fisher said that while the statistics given by Dr. Bryant were very interesting, the fact should be borne in mind that there are many sane people who are chronic sufferers from naso-pharyngeal and aural diseases of one kind or another, and a comparative table should be made before we could form a true estimate of the influence of these diseases as etiological factors in insanity.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

May 17, 1906.

The President, Dr. Tuttle, in the Chair.

Two Cases of Lead Poisoning.—Were reported by Dr. Baldwin, and the patients were shown.

Dr. Knapp had seen several cases of very severe and extensive lead paralysis in the past, in which the legs had been affected, and there was a well-marked toe-drop. Of late years, owing to better prophylaxis in lead factories and among painters, lead paralysis is less often seen, and such severe cases are very rare. The hands of Dr. Baldwin's patient suggest the condition sometimes seen in the early stages of lead paralysis, where the ring and middle fingers are dropped, but the patient can still extend the index and little fingers.

Brain Tumor.—Dr. Waterman showed a specimen of brain tumor which was of special interest on account of the relation of the site of the neoplasm to the character of the symptoms.

The patient was first seen four and a half years ago. She was at that time a girl of thirteen, and had suffered for several weeks from intense headache and persistent vomiting, and from gradual loss of vision. There was a double optic neuritis, but a careful examination disclosed no localizing signs of the tumor, which was apparently the cause of the symptoms.

She was given iodides and improved rapidly, regarding the pain and vomiting, but the loss of sight became nearly complete. Since that time, till recently, she has lived in comfort, though nearly blind, except that every six or eight months she was confined to her bed on account of severe headache and constant vomiting. Each of these spells lasted two or three